These authors have recently studied the toxicity of the normal sweat and have repeated the experiments with epileptic sweat. In the first series of ten cases the epileptic sweat exhibited no difference in comparison with normal sweat, save that the digestive tube was more affected than in the latter case, while the urine was also more frequently voided. Fatalities were not more commonly produced. In a second series, however, the epileptic sweat proved fatal in every one of eight cases. and in as small amounts as 137 c. c., 151 c. c., etc. It was exceptional, however, for convulsions to develop. The autopsy findings showed much inconstancy. To explain such radical differences in results in these two groups of cases, the authors state that in the first series, the sweat used was interparoxysmal, while in the second series it was taken either at the onset of the attack or very soon afterward.

To sum up, the interparoxysmal sweat is normal sweat, and the paroxysmal sweat is toxic and lethal in action. The toxicity is soon lost after the attack. This toxic property of sweat is genuine and not due to want of isotony with the blood serum of the rabbit. For that matter, the isotony is often almost perfect. Thus freezing point of the paroxysmal sweat runs from 40 to 56, while the interparoxysmal sweat is about 40.

Referring to the prior article by these authors on the normal sweat (Compes Rend. Soc. de Biol., Nov. 23, 1900), a description for their method of collecting the sweat is given. The patient is placed in a coffin-shaped oven, his head alone being outside of the apparatus. The latter is heated by a current of hot air; the skin was previously ascepticised. The sweat during the baking process ran down into a trough whence it gravitated to a sterile collecting vessel. In some cases the authors collected as much as 800 c. cm. of sweat from a single individual. CLARK.

UNFALL UND EPILEPSIE (Epilepsy and Accident). F. Bahr (Monatssch. f. Unfallheilkunde, Oct. 15, 1900).

The literature of traumatic epilepsy of peripheral origin is a meager one. Cases are confined almost wholly to the results of direct injury to the skull and brain. There is hardly anything on the subject of epilepsy as a result of peripheral wounds. In the Franco-German war, out of 77,461 wounded (not including cases which resulted fatally) there were 17 cases of traumatic reflex epilepsy. Despite this infrequency the author believes it well to call attention to the subject. He has been able to get notes of three cases in which an unmistakeable connection can be shown between accident and epilepsy.

Case I. Male, aged 44; his history negative. He wrenched his right shoulder while carrying a drain pipe. The clavicle of the same side also was fractured; the whole right half of thorax was sensitive. Traumatic hemoptysis. After his injuries had healed, he complained of spells of forgetfulness, unconsciousness, vertigo, etc. About a year after the injury, he developed typical epileptic attacks. Case II. Male, aged 44; history before injury was quite negative. His accident consisted of a fall which apparently caused a contusion of the lung. He became subject to epileptic attacks. Case III. Male, aged 56; his history was negative; he had a fall from a ladder. He developed epileptic attacks which appeared to be called forth by injury to the left lower extremity.

All these cases were in middle-aged men; it is rare for epilepsy to begin after the age of 20 years. The coincidence is a much more plausible explanation than a reflex epilepsy of traumatic peripheral origin, cases of which occur much less frequently the more carefully epilepsy is studied. CLARK.

PATHOLOGY.

PATHOLOGY OF HERPES ZOSTER. C. Head and P. Campbell (Brain, Autumn Number, 1900).

These authors have made an extensive series of observations on the pathology of herpes zoster, founded on cases where the patient died at various periods subsequently. The acute changes found in the posterior root ganglion of the nerve supplying the affected area of the skin, consist of (1) an extensive acute inflammation with the exudation of small round deeply-staining cells; (2) extravasation of blood; (3) destruction of ganglion cells and fibers; (4) inflammation of the sheath of the ganglion. If severe, such a condition leaves a scar in that part of the ganglion affected and leads to thickening of the sheath above the affected area. On the other hand if the eruption has not been severe, all traces of the inflammation present in the acute stage may pass away, leaving the ganglion apparently normal. The changes in the posterior nerve root corresponded to the results which might have been expected from the lesion of the ganglion; they consisted of an acute degeneration followed by a greater or less amount of secondary sclerosis according to the severity of the acute destruction. The anterior root was in all cases normal. In the mixed peripheral nerves degeneration likewise occurs and can be traced right up to the fine twigs which pass into the skin to supply the area over which the eruption is distributed. The time relations of the degeneration and subsequent sclerosis to the eruption was the same in the peripheral nerve and the posterior root. The degeneration in the posterior roots can be traced into the posterior columns of the spinal cord. Where the eruption extends on to the arm the degenerated fibers can be followed in the cord from the root zone to the postero-external column, and by this path up to the nucleus cuneatus. Where the eruption is on the leg the field of degeneration passes into the postero-median column. The posterior roots in the dorsal region, containing as they do afferent fibers from the trunk, consist mainly of short fibers which do not not form a part of either the postero-internal or the postero-external columns in the cervical region of the spinal cord, but run up in the root zone adjacent to the posterior horn, get less in number as they ascend and disappear in a variable (6 to 16) number of segments. Thus the long fibers which form the postero-internal and postero-external columns in the cervical region come almost exclusively from the leg and arm respectively. Zoster of the branches of the trigeminal is associated with a similar lesion in the Gasserian ganglion to that found in the posterior root ganglia in cases of zoster of the trunk and limbs. This lesion causes secondary degeneration in the sensory root of the Gasserian ganglion both in its extra- and intra-medullary course. Zoster in all respects resembling that arising spontaneously may be produced by implication of a posterior root ganglion in inflammatory processes secondary to malignant disease, tubercle, or injury.

Sections through an unbroken vesicle of herpes zoster show a cavity, the floor of which is formed of naked papillæ which are in a condition of profound inflammation and are infiltrated with small